Intraoperative low-tidal-volume ventilation

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Intraoperative Low-Tidal-Volume Ventilation

TO THE EDITOR: We are troubled by the ventilation strategy selected for the control group (or nonprotective-ventilation group) in the study by Futier et al. (Aug. 1 issue).[1] This strategy (nonprotective ventilation with a tidal volume of 10 to 12 ml per kilogram of predicted body weight, with no positive end-expiratory pressure [PEEP] and no recruitment maneuvers) is known to be potentially harmful and is outdated (the authors cite a study from 1963[2] to define their standard of care). The tidal volumes recommended in contemporary strategies[3,4] for perioperative ventilation are less than 10 ml per kilogram of predicted body weight, and they are provided with PEEP.

To analyze practice patterns for patients undergoing anesthesia, we undertook a study in which we reviewed data from 230,386 surgical procedures at two institutions. These data show that PEEP with a median of 5 cm of water was used in 60.4% of procedures. Median tidal volumes declined from 9.2 to 7.9 ml per kilogram of predicted body weight between 2005 and 2013.

Prospective studies such as that by Futier et al. are most relevant if they derive their data from recent observational studies that used contemporary standards of care. Moreover, these data can be derived from clinical-decision support databases.[5] Do the authors have contemporary data to support their choice of ventilation strategies?

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TO THE EDITOR: Futier et al. attributed the outcome of fewer postoperative complications in the protective-ventilation group, in which low-tidal-volume ventilation and PEEP were used, mainly to the prevention of atelectasis. We postulate that microaspiration could be another reason for the higher rate of postoperative pneumonia in the nonprotective-ventilation group.

In our recent study,[1] we found that without the provision of PEEP there was downward leakage of fluid across the cuff of the endotracheal tube, even at a recommended cuff pressure of 20 to 30 cm of water. This leakage was eliminated with the use of a PEEP as low as 5 cm of water or with the use of endotracheal tubes with newer cuff designs. In another study[2] in which high-tidal-volume ventilation was compared with low-tidal-volume ventilation, with a PEEP of 5 cm of water in both groups during major upper abdominal surgery, no significant difference was detected in postoperative lung function or clinical outcome. We believe the application of PEEP is the critical variable in the study by Futier et al. We also wonder whether the inflammatory condition associated with pneumonia contributed to the anastomotic leak.

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No potential conflict of interest relevant to this letter was reported.

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TO THE EDITOR: Futier et al. compared two approaches to ventilation during laparoscopic and open surgery (laparotomy). Primary outcomes were recorded in 69 of the 85 patients undergoing laparoscopic surgery and in 7 of 315 patients undergoing nonlaparoscopic open surgery. The primary strategies used in open and laparoscopic surgical ventilation support are different, partly because of the insufflation of carbon dioxide that occurs during laparoscopic surgery.[1-4] To avoid hypercapnia and respiratory acidosis during laparoscopic surgery, it is common to use gradually increasing tidal volumes, ultimately leading to the use of high-volume–high-pressure mechanical ventilation during prolonged laparoscopic surgeries. Tidal volumes can be adjusted by monitoring the end-tidal carbon dioxide concentration and the acid–base status of the blood. To what extent were the ventilator settings changed during laparoscopic surgery to meet its unique needs? Can the lack of such adjustments explain the observed outcomes?

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THE AUTHORS REPLY: In response to Wanderer et al.: we fully concur that there has been a gradual decrease over time in the use of high-tidal-volume ventilation and that the ventilation strategy used in our control group may therefore not fully reflect practice in all hospitals. Nevertheless, lung protection is often incorrectly reduced to lowering tidal volume, with either very low levels of PEEP (<5 cm of water) or no PEEP. Low-tidal-volume ventilation alone is not only ineffective[1] but also may be deleterious. Contemporary data on the use of PEEP in addition to lower tidal volume are scarce. A recent analysis of an electronic database from the University of Colorado suggested that a combination of a tidal volume of more than 8 ml per kilogram of predicted body weight and a PEEP of less than 5 cm of water represented the standard of care in more than 60% of patients.[2] Data from our observational study indicate that regardless of the PEEP level used, a tidal volume in the range of our protective-ventilation strategy (6 to 8 ml of water per kilogram of predicted body weight) was used in 30.1% of patients.[3]

We fully agree with the comment by Lam et al. on the pivotal role of PEEP in addition to lower tidal volume in the protective-ventilation strategy. However, the additional contribution of recruitment maneuvers (which were a part of this strategy) should not be underestimated, since PEEP levels in the range of those used in our experimental conditions are not effective in reopening nonaerated lung regions.[4] Further investigation will be required to address the question of whether the inflammatory conditions associated with the development of pneumonia or the use of mechanical ventilation can explain anastomotic leak.

Mynbaev et al. ask about the possible influence of laparoscopic surgery on the occurrence of the primary outcome. Data presented for laparoscopic surgery in Table S1 of the Supplementary Appendix of our article (available at NEJM.org) were erroneously interchanged with the data for nonlaparoscopic surgery. The primary outcome measure should have been shown for 10 of the 85 patients who underwent laparoscopic surgery rather than for 69 of those 85 patients (odds ratio, 0.50; 95% confidence interval, 0.25 to 1.03; P=0.06) and for 66 of the 315 patients who underwent nonlaparoscopic surgery. We regret the error; a corrected version of the Supplementary Appendix is available at NEJM.org.

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Glucose Levels and Risk of Dementia

TO THE EDITOR: Crane and coworkers (Aug. 8 issue)[1] report on the relationship between glucose levels and the risk of dementia in participants whose mean age at baseline was 76 years. Their analysis is limited by the fact that the established and readily available confounder of renal function had not been accounted for. In the United States, 62.2% of persons 80 years of age or older have an estimated glomerular filtration rate (GFR) of less than 60 ml per minute per 1.73 m^2 of body-surface area.[2] Moderate renal impairment has been shown to be associated with an excess risk of incident dementia among persons in good-to-excellent health.[3] A community-based cross-sectional study showed that global performance and specific cognitive functions are negatively affected early in chronic kidney disease.[4] In a community-based study with a follow-up period similar to that in the study of Crane et al., the change in renal function over time was related to the change observed in global cognitive ability, verbal episodic memory, and abstract reasoning.[5] Aside from the estimated GFR, increased albuminuria is also independently associated with a faster decline in cognitive function. Hence, renal function is an important factor contributing to cognitive impairment and cognitive decline.

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No potential conflict of interest relevant to this letter was reported.

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THE AUTHORS REPLY: Kielstein raises the possibility that renal function may confound the association that we found between glucose levels and dementia risk. We are aware of the literature that Kielstein references. Indeed, these data pro-